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COGNITIVE DISORDERS AFTER A STROKE

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Introduction. As medical care for stroke patients improves and mortality decreases, the proportion of patients with cognitive disorders will increase. An important factor contributing to the increase in the number of this category of patients is the change in The demographic situation in the world is an increase in the number of elderly and senile people. In elderly people, even a small ischemic or hemorrhagic stroke can worsen the existing slight decrease in cognitive functions associated with age-related changes, chronic cerebral circulatory insufficiency, hidden by the current neurodegenerative process. At the same time, cognitive decline worsens the quality of life of patients, leads to impaired social activity and disability.

Key words: cognitive impairment, ischemic stroke, vascular pathology, memory impairment, dementia.

Vascular cognitive impairment ranks 3rd in prevalence after dementia in the disease Alzheimer's disease (AD) and mixed dementia. 6 months after a stroke, moderate cognitive impairment (MCI) is diagnosed in 45-80% of patients, dementia in 10-15% [2, 3]. After 5 years, dementia develops in 20-25% of patients. The risk of developing dementia after stroke in patients over 60 years of age in the first 3 months is 9 times higher than in the control group [4]. The average life expectancy of patients with vascular dementia is about 5 years, which is less than the life expectancy of patients with asthma [5]. Dementia significantly increases the risk of recurrent stroke and death from cardiovascular diseases [6].

The development of post-stroke cognitive disorders can be discussed if there is a clear temporal link between stroke and cognitive decline. functions. Usually, post-stroke cognitive impairments develop in the first 3 months after a stroke (early post-stroke cognitive decline), but no later than 12 months (late cognitive decline). A three-month interval is one of the criteria for vascular dementia NINDS-AIREN [7].

Patients with damage to the dominant hemisphere have a higher risk of cognitive impairment. Thus, according to A.N. Bogolepova [8], circulatory disorders in the left carotid system were accompanied by cognitive decline in 46% of cases, circulatory disorders in in the right carotid artery system – in 15%, in the vertebrobasilar system – in 8% of cases. Additional

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factors that increase the risk of developing post-stroke dementia are low levels of education, long-term hypertension, diabetes mellitus, heart disease (atrial fibrillation, heart failure), additional cerebral pathology (chronic cerebral circulatory insufficiency, atrophic changes in the brain) [9].

Cognitive decline after a stroke may be based on the following causes.

-A heart attack in the strategic area of the brain that plays the largest role in the regulation of mental functions (thalamus, striatum, prefrontal frontal cortex, hippocampus, angular gyrus). The incidence of heart attacks in strategically important areas is approximately 5%. In this case, cognitive impairments appear suddenly, followed by a stable course or a slight regression of symptoms. The nature of neuropsychological disorders is diverse and depends on the location of the lesion. Thus, damage to the thalamus is accompanied by the development of spontaneity., apathy, adynamia, slowness of mental processes, impaired memory for current events, decreased concentration, increased drowsiness. The cognitive defect is explained by secondary frontal dysfunction as a result of impaired thalamocortical connections. When the thalamus of the dominant hemisphere is affected, thalamic aphasia is associated with a large number of paraphasias, but with a preserved understanding of spoken speech and no difficulty in repeating phrases for the doctor. Stroke in the striatum is characterized by a combination of neurodynamic and regulatory disorders resembling the subcortical variant of vascular cognitive disorders. Stroke in the prefrontal areas of the frontal cortex is accompanied by the formation of apaticoabulous syndrome with a decrease in criticism of one's condition, perseverations, and echolalia. Damage to the angular gyrus (occipito-parietal-temporal junction) is characterized by the development of visual-spatial agnosia, constructive apraxia, acalculia, and semantic aphasia.

- Multiinfarction brain damage. This condition is a consequence of large territorial infarcts of cortical or cortical-subcortical localization. A multiinfarction lesion is caused by thrombosis or embolism of large cerebral vessels. The loss of more than 50 mm3 of brain matter is necessary for the development of dementia. Cognitive disorders include operational disorders associated with damage to the cortical sections of various analyzers, corresponding to the localization of heart attacks. Along with this, various focal neurological disorders are observed.

-Cognitive impairment due to hypoperfusion of the brain. They develop in pathology of central hemodynamics, when there is a sharp decrease in perfusion pressure in the brain. In this case, infarcts of varying volume are formed in the area of adjacent blood circulation, at the junction of vascular basins. The development of such infarcts is largely determined by the capabilities of collateral circulation, the preservation of autoregulation of cerebral blood flow, which is often disrupted by hypertension. The severity and nature of cognitive disorders depend on the location and degree of brain damage.

- A combination of heart attacks with diffuse damage to the white matter. The development of cognitive disorders is based on damage to small vessels of the brain – microangiopathy on the background of hypertension. The penetrating arteries, which supply blood to the subcortical ganglia area and subcortical white matter, are subject to the greatest changes in hypertension. These arteries belong to terminal-type vessels, i.e. they have practically no

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collaterals, and their vascularization zone is most vulnerable in regarding ischemia against the background of fluctuations in systemic hemodynamics. The lesion of white matter is characterized by a decrease in its density – leukoareosis, expansion of perivascular spaces, demyelination, gliosis. First, these changes appear near the anterior horns of the lateral ventricles, and then spread caudally. Subsequently, subcortical leukoarrhoea joins periventricular leukoarrhoea. Lacunar infarcts resulting from occlusion of small vessels may manifest as clinical changes characteristic of stroke, but are most often clinically mute. The predominant localization of lacunae is the shell, caudate nucleus, thalamus, corona radiata, and bridge of the brain. The clinical picture of cognitive decline in this case is characterized by gradual progression with impaired executive functions with slow mental processes, decreased concentration, and thinking flexibility., the ability to analyze information, identify similarities and differences. At the same time, memory impairments are moderate in nature and are associated with difficulty in extracting information while maintaining its storage and recognition. A feature of cognitive disorders of this localization is their frequent combination with emotional-affective disorders and gait disorders such as frontal dysbasia.

- A combination of vascular brain damage with neurodegenerative changes. The frequency of a combination of asthma and vascular dementia increases with age. According to pathomorphological studies, only in 40% of cases post-stroke dementia develops directly due to vascular causes. The remaining cases are classified as mixed versions. Local vascular brain damage can generally increase the total volume of brain damage and contribute to the clinical manifestation of neurodegenerative disease. In other cases In situations with latent asthma, a small lacunar stroke in a strategically important area, which by itself cannot cause a decrease in cognitive functions, provokes an increase in cognitive deficit.

A characteristic feature of post-stroke cognitive disorders is a violation of the regulation of voluntary activity (decreased motivation, flexibility of thinking, impaired planning, reaction speed, concentration of attention) associated with dysfunction of the frontal lobes with a non-severe memory defect. As a rule, cognitive The deficiency is combined with focal neurological symptoms depending on the location of the lesion, as well as with changes in mood background and emotional lability. A prerequisite for the diagnosis of vascular cognitive disorders is the detection of changes in magnetic resonance imaging/computed tomography (MRI/CT).

The degree of cognitive impairment after a stroke can range from mild cognitive impairment to severe dementia. In contrast to the steadily progressing process in asthma, post-stroke cognitive impairments can be reversible. Therefore, timely The detection of mild cognitive impairment and the correction of treatment can stabilize the process for a long time. The most important difference between the mild cognitive impairment stage and dementia is the preservation of the main types of daily activity (social, household independence), as well as criticism of one's condition.

The improvement of cognitive functions after a stroke is largely explained by the phenomena of neuroplasticity, i.e. the ability of nervous tissue to rebuild due to the involvement of previously inactive but functionally close areas, the reorganization of pathways and interneuronal connections, as well as collateral springing of preserved cells with the formation of new synapses. The stimulation of neuroplasticity processes is provided by the

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activation of neurometabolic processes, the release of neurotrophic factors, as well as by improving the functioning of neurovascular units due to changes in the reactivity of small vessels. Therefore, the strategy for the treatment of vascular cognitive disorders should include several areas: improving brain perfusion, the use of neuroprotective agents, as well as drugs that stimulate metabolism. neurotransmitters (dopamine, norepinephrine, acetylcholine) involved in cognitive processes. Neurotransmitter drugs have a symptomatic effect in vascular cognitive disorders – they improve attention, reaction speed, memory, and speech functions. In controlled studies in dementia, the effectiveness of acetylcholinesterase inhibitors (donepezil, rivastigmine, galantamine), as well as the antiglutamatergic drug memantine, has been proven. Correction of risk factors is of great importance. (primarily hypertension, hyperlipidemia) and prevention of recurrent vascular episodes (using antiplatelet agents, anticoaculants).

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